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<u>L12</u>	(cathelicidin\$) and (stimulat\$ or enhanc\$) same (monocyt\$ or apc\$ or antigen\$ or macrophag\$)	9	<u>L12</u>
<u>L11</u>	(cathelicidin\$)same (stimulat\$ or enhanc\$) and (monocyt\$ or apc\$ or antigen\$ or macrophag\$)	0	<u>L11</u>
<u>L10</u>	(cathelicidin\$)same (stimulat\$ or enhanc\$)same (monocyt\$ or apc\$ or antigen\$ or macrophag\$)	0	<u>L10</u>
<u>L9</u>	(cathelicidin\$)same (adjuvant\$ or vaccin\$ or immunogen\$)	0	<u>L9</u>
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<i>DB=PGPB,USPT; PLUR=YES; OP=ADJ</i>			
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<u>L3</u>	(camelid or cammelid) and tnf\$	62	<u>L3</u>
<u>L2</u>	s (camelid or cammelid) and tnf\$	0	<u>L2</u>
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L12: Entry 1 of 9

File: USPT

May 10, 2005

DOCUMENT-IDENTIFIER: US 6890537 B2

TITLE: Antimicrobial theta defensins and methods of using same

Detailed Description Text (59):

The invention additionally provides a method of reducing or inhibiting growth or survival of a microorganism in an individual by administering a molecule, wherein the molecule increases expression of a theta defensin. Theta defensins are polypeptides expressed in leukocytes of mammals, in particular primates, including humans. Thus, theta defensins function as part of the endogenous defense system for a mammal to combat microbial infections. Since theta defensins are expressed in mammals, methods to increase expression of theta defensin in the organism can be used to reduce or inhibit microbial growth in the organism. Using the genomic clones described herein, one skilled in the art can readily determine regulatory molecules that can alter transcription of a theta defensin gene and screen for those molecules that effect an increase in theta defensin expression. Cytokines, for example, monocyte chemoattractant protein 1 (MCP-1), interleukin 8 (IL8) or other cytokines, that activate granulocytes can be tested for stimulatory activity of theta defensin expression. Cytokines, or other compounds, can be screened for stimulatory activity. Compounds having stimulatory activity can be used to increase expression of a theta defensin in an organism to reduce or inhibit growth or survival of a microorganism in an individual.

Detailed Description Text (75):

Searches for amino acid sequence similarity to RTD-1 were carried out using all 18 possible linearized peptides as query sequences (Altschul et al., Nucleic Acids Res. 25:3389-3402 (1997)). Taking into consideration the linear cysteine spacing and disulfide connectivities of RTD-1, the most similar protein sequence identified was that of the porcine antimicrobial peptide protegrin 3 (PG-3) (see FIG. 4). Protegrins are 17-18 amino acid, di-disulfide containing peptides that are members of the cathelicidin family of antimicrobial peptides (Zanetti et al., FEBS Lett. 374:1-5 (1995)). Cathelicidins share a high degree of sequence similarity in the prepro-regions of their precursors, but the carboxyl termini, containing the antimicrobial peptide segments, vary markedly. Like protegrins, RTD-1 is predicted to be predominantly composed of two disulfide stabilized .beta. strands connected by turns.

Detailed Description Text (77):

As shown in FIG. 4, RTD-1 is remarkably similar to the solution structure of protegrin 1. This similarity suggested the possibility that RTD-1 is a member of the cathelicidin family. However, subsequent studies demonstrated that RTD-1 is not a cathelicidin, but rather the product of two .alpha.-defensin-related genes (see Example V).

Other Reference Publication (15):

Zanetti et al., "Cathelicidins: a novel protein family with a common proregion and a variable C-terminal antimicrobial domain," FEBS Lett., 347:1-5 (1995).

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L12: Entry 4 of 9

File: USPT

Oct 12, 2004

DOCUMENT-IDENTIFIER: US 6803044 B1

TITLE: Antimicrobial and anti-inflammatory peptides for use in human immunodeficiency virus

Brief Summary Text (10):

S. aureus enterotoxins are associated with food poisoning and appear to act by stimulating emetic receptors in the abdominal viscera to cause vomiting and diarrhea. In addition, S. aureus enterotoxins are superantigens. They bind to macrophage major histocompatibility complex (MHC) class II molecules at a conserved site away from the hypervariable groove and then to the site of the T-cell receptor beta chain, rather than to its variable face that recognizes conventionally processed antigens bound to the MHC. This leads to massive stimulation of host T cells and release of cytokines, which mediate the systemic effects of S. aureus enterotoxin.

Detailed Description Text (3):

.alpha.-MSH (SYSMEHFRWGKPV) (SEQ ID NO: 3) is a pro-opiomelanocortin (POMC)-derived tridecapeptide with potent antipyretic and anti-inflammatory influences. The C-terminal tripeptide of .alpha.-MSH, KPV exerts anti-inflammatory effects in vitro and in animal models of inflammation, that are similar to those of the entire 1-13 sequence. Therefore, KPV is considered the anti-inflammatory "message sequence" of the peptide. Five G-protein-linked melanocortin receptors (MC-1R through MC-5R) have been isolated and cloned. They bind .alpha.-MSH and other melanocortin peptides such as ACTH with different affinities; MC-R are expressed in the brain and in peripheral tissues. The precursor of .alpha.-MSH, POMC, is widely expressed in tissues. However, there are distinct regional differences in expression and post-translational processing and certain cells produce, constitutively or under appropriate stimulation, greater amounts of .alpha.-MSH. .alpha.-MSH occurs in high concentrations in barrier organs such as the gut and the skin. Activated macrophages and microglia likewise produce substantial amounts of .alpha.-MSH and there is evidence that the peptide has an autocrine anti-inflammatory influence in these cells that express melanocortin receptors. The anti-inflammatory effects of .alpha.-MSH are exerted partly via inhibition of certain inflammatory mediators, such as cytokines and nitric oxide, likely through inhibition of the transcription factor NF-.kappa.B.

Detailed Description Text (10):

The chronically HIV-1 infected promonocytic U1 cell line was maintained in complete culture medium (RPMI 1640 supplemented with 10 mM Hepes), 2 mM L-glutamine (Sigma-Aldrich), 10% heat-inactivated FCS (HyClone Laboratories, Logan, Utah, USA), penicillin at 100 units/mL and streptomycin at 100 .mu.g/mL (Gibco Laboratories, Grand Island, N.Y.) in log phase of growth. Pilot experiments were performed to determine optimal cell density, stimuli concentration, and kinetics of HIV-1 p24 antigen production using our culture conditions. Before use, cells were washed three times with HBSS (Gibco) to remove extracellular virus. Cells were plated onto 24-well flat-bottomed plates at a concentration of 2.times.10⁵/mL (final volume 1 mL) with medium alone or plus TNF-.alpha. (10 ng/mL (R&D Systems; Oxford, England, UK) in the presence or absence of .alpha.-MSH [1-13] or KPV in concentrations from 10.sup.-13 to 10.sup.-4 M. In further experiments, KPV was added in the 10.sup.-5 M concentration to U1 cells stimulated with TNF-.alpha. (10 ng/mL), IL-6 (20 ng/mL), IL-10 (20 ng/mL (R&D Systems) or PMA (1 ng/mL) (Sigma-Aldrich Chemicals, St.

Louis, Mo., USA). Supernatants were removed by centrifugation after 48 hr incubation at 37.degree. C. in 5% CO.sub.2, and stored at -80.degree. C. In crowding experiments, U1 cells were seeded at the density of 2.times.10.sup.5 mL and maintained in culture at 37.degree. C. in 5% CO.sub.2 without change of medium for 7 days. KPV (10-5M), or an equal volume of medium, were added on day 1. In all experiments each condition was tested in triplicate.

Detailed Description Text (29):

.alpha.-MSH receptor MC-1R gene expression was determined in resting and PMA-stimulated U1 cells. In both conditions, a PCR product specific for MC-1R with the expected length of 416 bp was detected (FIG. 1). Supernatants of resting and stimulated U1 cells were analyzed for production of .alpha.-MSH. There was a small but consistent production of peptide after 48 h culture in unstimulated conditions (5.2.+-.0.3 pmol/mL). When cells were coincubated with PMA, .alpha.-MSH in the supernatants was increased to 12.90.+-.0.42 pmol/mL. To determine effects of blockade of endogenous .alpha.-MSH on HIV-replication, the peptide was immunoneutralized with a specific anti-.alpha.-MSH antibody. p24 antigen was measured in the supernatants from resting cells and from those exposed to PMA or in crowding conditions. In cells incubated with the anti-.alpha.-MSH antibody there was a substantial increase in p24 release under unstimulated and crowding conditions and after stimulation with PMA (FIG. 2). The irrelevant IgG did not alter p24 release in any condition.

Detailed Description Text (40):

The presumed autocrine circuit for control of viral replication may be similar to that found in related studies. Activity of macrophages and glial cells can be modulated via an endogenous autocrine circuit that depends upon .alpha.-MSH and specific melanocortin receptors. Murine and human macrophages contain MRNA for the melanocortin receptor MC-1R and they secrete .alpha.-MSH. Blockade of endogenous .alpha.-MSH by immunoneutralization increased production of proinflammatory cytokines and nitric oxide in microglia. Incubation of resting macrophages with antibody to MC-1R promoted TNF-.alpha. production. Further, immunoneutralization of MC-1R markedly reduced the inhibitory influence of .alpha.-MSH on TNF-.alpha. production by activated macrophages. The present experiments in HIV-infected cells indicate that an autocrine circuit based on .alpha.-MSH likewise occurs in infected monocytes: U1 cells expressed MC-1R and produced .alpha.-MSH, and blockade of the endogenous peptide significantly enhanced HIV expression in both resting and stimulated cells. This suggests that endogenous .alpha.-MSH reduces viral expression via an autocrine mechanism. Such antiviral influences based on .alpha.-MSH could be significant to host protection. That is, in peripheral and central phagocytes, which are the main reservoir of the virus production, the action of .alpha.-MSH could reduce viral burden.

Detailed Description Text (42):

Replication of HIV is dependent on the state of activation of infected cells and is regulated by interactions between viral and host factors. Among the latter, proinflammatory cytokines have a prominent enhancing effect on HIV replication. TNF-.alpha. and other cytokines such as IL-1 and IL-6 promote HIV replication and have detrimental influences on HIV disease progression. Inhibition of such proinflammatory cytokines is, therefore, a target for adjunctive therapies of HIV infection. Endotoxin-stimulated production of IL-1, IL-6, and TNF-.alpha. in whole blood of HIV-positive patients was substantially reduced by .alpha.-MSH. Further, .alpha.-MSH inhibited TNF-.alpha. production by peripheral blood mononuclear cells stimulated with HIV envelope glycoprotein gp 120. .alpha.-MSH also reduced brain TNF-.alpha. in an in vivo model of brain inflammation. In addition to these cytokines, .alpha.-MSH inhibited several products of activated macrophages including neopterin and nitric oxide. The present results in chronically infected UI cells indicate that .alpha.-MSH peptides inhibit HIV replication induced by major stimuli known to up-regulate the virus in this cell model of chronic HIV infection.

Detailed Description Text (94):

It is known that .alpha.-MSH shares a number of similarities with other natural antimicrobial peptides such as the defensins or the cathelicidins:

Other Reference Publication (67):

Domk-Optiz, I., et al., "Stimulation of Macrophages by Endotoxin Results in the Reactivation of a Persistent Herpes Simplex Virus Infection," Scand J. Immunol. 32 (2):69-75 (1990).

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